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DISEASES OF THE NERVOUS SYSTEM

A PRACTICAL JOURNAL ON PSYCHIATRY AND NEUROLOGY

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DISEASES of the NERVOUS SYSTEM

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A PRACTICAL JOURNAL ON PSYCHIATRY AND NEUROLOGY

*The Anterior Perforating Arterioles and Their Relation to the Internal Capsule**

ARTHUR F. YOUNG, M.D. and LOUIS J. KARNOSH, M.D.
Cleveland, Ohio

Despite the impressive and painstaking studies of cerebral circulation made in recent years, the vascular supply of the internal capsule remains to many clinicians a subject of considerable confusion and vague delineation. Possibly one reason for this is the matter of inept terminology. The blood vessels which nourish the internal capsule are usually presented or identified in their relationship to the basal ganglia and to various other brain stem structures, most of them being designated as striatal arterioles. The majority of textbooks dispose of them by the erroneous or misleading term "lenticulo-striate arteries."

Actually the arterioles in question supply blood to several and varied structures in the cerebral hemispheres. Alexander[1] has correctly denominated most of them the "striatocapsular arterioles" and to the longest and most complicated one of this group he has applied the cumbersome term, "the pallidohippocampo-capsular artery." Even this does not encompass the complete role of this blood vessel, which is familiarly known as the anterior choroidal artery, for it provides a considerable blood supply to the external geniculate body, to the crus cerebri and to the upper portion of the choroid plexus of the lateral ventricles.

If for no other reason, and whatever other structures they may supply, these most vulnerable of all cerebral blood vessels should be grouped together because of the pathology they so frequently impose upon the internal capsule. One method of avoiding confusion would be to discontinue terminology based upon partial distribution and call them all anterior perforating arterioles. They all possess the common characteristic of being relatively small vessels emerging at right angles from relatively large parent arteries and penetrating the brain in or near the anterior perforated substance. Upon these features so well presented in the demonstrations by Alexander are based the concepts of the propensity of these arterioles to miliary aneurysms, thrombosis and hemorrhage.

To trace these arterioles, to learn their ultimate distribution and to determine what, if any, anastomotic communications may exist with other circulations has been intriguing to anatomists, physiologists, and neuropathologists. The methods of investigation have ranged from careful manual dissection such as that carried out by Kolisko,[2] Beevor,[3] Aitken and Ayer[4] to the injection of the cerebral circulation with various materials such as metals or colloidon with subsequent digestion of the brain substance so as to leave a cast of the vascular tree.

Perhaps the most illuminating studies as far as the perforating arterioles to the internal capsule are concerned have been those of Abbie[5] and Alexander.[1] Both of these workers used India ink injection, the latter

*From the Department of Neurology of Cleveland City Hospital and the Department of Neuropsychiatry of the Cleveland Clinic Foundation.

combining it in a solution with formalin. The limitations of such methods of examination are readily recognized. First and foremost the nature of the injection material is such that it readily traverses not only the terminal ramification of the vessel to be investigated but the capillary network and venous system as well. Secondly, the India ink is itself a dye substance and its presence precludes an accurate histological examination of the tissues injected. Thirdly, the fluid nature of the injection substance permits easy extravasation and consequent distortion of surrounding tissues as well as those to be studied. Alexander and his co-workers[6] endeavored to circumvent certain of these disadvantages by further studies with the use of benzidine stain. This method, however, while readily demonstrating vascular channels, does not permit identification of the source of supply of these channels or their ultimate terminations.

The publication by Schlesinger[7] of his excellent technique to facilitate study of the coronary arteries by the injection of his radio-opaque material and subsequent roentgenographic recordings opened a new approach to the scrutiny of cerebral circulation at post-mortem. The adaptation of the Schlesinger technique using a barium-latex injection mass as described by Salans and Tweed[8] suggested this particular investigation of the anterior perforating arterioles.

In this study the injection apparatus of Salans and Tweed[8] was utilized, but the barium-latex injection mass was modified in the following particulars. The particle size of the injection mass† has been standardized at fourteen micra by screenings, viscosity determinations and ocular micrometer examinations, so that while penetrating to vessels of pre-capillary diameter, the capillary net work, itself, is not injected. Such an injection stopping just short of the capillary bed allows a clear demonstration of the arteriolar tree unobscured by capillaries or venous channels. The injection mass is quickly and permanently hardened by placing the specimen in a ten per cent solution of formo-saline after the injection is completed. Subsequent dissections may then be carried out without extravasation of any of the injected material. The injection mass is available in several different colors and is color-fast, an advantage which permits of the use of several colors in the same specimen, and of easy identification of the vessels to be studied not only during gross dissection but also in the histological preparations.

The technique currently used is as follows: The brain is removed at autopsy in toto, leaving a 2 cm. portion of the spinal cord attached and being careful not to tear or lacerate the surface of the brain. Cannulae consisting of a specially modified No. 20 needle are then inserted into the specific vessels to be investigated and securely fastened in place by tying a loop of thread behind the projecting collar of the needle. The specimen is then perfused with normal saline for a period of five minutes at a pressure of 120 mm. of mercury. At the end of this period the perfusion is stopped and the cannulae are connected to the various bottles of injection material. The barium-latex compound is injected for a period of ten minutes at a pressure of 120 mm. of mercury. With cannulae in place but disconnected from the injection apparatus, the specimen is placed in a jar of formo-saline until fixation of the brain tissue is complete after which time both injection mass and brain material are adequately hardened and ready for dissection studies or microscopic section.

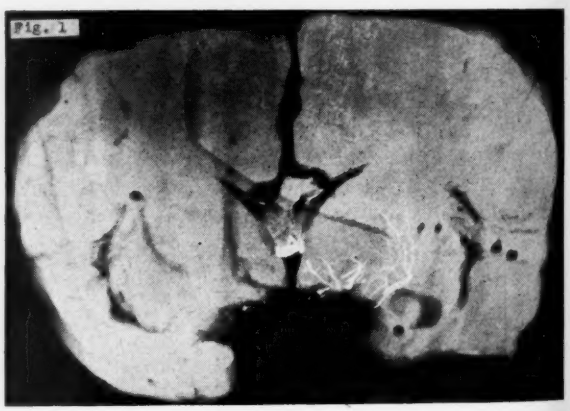

Figure 1: Roentgenograph through a coronal slice of the brain in the region of the anterior pillar of the fornix. The injection of the radio-opaque material was made only into the perforating arterioles of the middle cerebral artery. The neo-striatum is richly vascularized. No pre-capillary vessels are seen in the anterior limb of the internal capsule except those which traverse it to supply the caudate nucleus.

†A suspension of barium sulfate in an aqueous ammoniated solution of liquid latex is used. Compound No. 60-22724A (red), No. 60-22724B (blue) and No. 60-24899 (white) obtained from American Anode Company, Akron, Ohio.

In preparing the specimens for special study of the anterior perforating arterioles, the injections were made into the stump of the middle cerebral artery with ligatures blocking off all branches other than those entering the anterior perforated space, in the same manner as utilized by Alexander. This, of course, does not include all the striato-capsular arterioles, for in most brains two or more branch off the anterior cerebral artery and in a small proportion of specimens Alexander found a so-called capsular artery arising directly from the internal carotid.

In Figure 1 is a demonstration of the course, distribution and structure of the perforating arterioles arising from the right middle cerebral artery. This is a roentgenograph of a coronal slice through the anterior limb of the internal capsule and the anterior pillar of the fornix. The specimen demonstrates how well the radio-opaque injection mass is restricted to the arteries and arterioles.

The outermost perforating arterioles of the middle cerebral artery after entering the perforated substance are seen passing laterally and then forming an angle to direct their course in a dorso-medial direction so as to conform to the lamellations of the corpus striatum. There appears to be considerable anastomosis and branching within the substance of the putamen, but as the arterioles approach the internal capsule, such communications completely disappear. Vessels of true arteriolar calibre cross the internal capsule only to enter the head of the caudate nucleus. No anastomoses are demonstrated in the substance of the internal capsule itself.

The more proximal arterioles entering the perforated space from the middle cerebral artery are definitely shorter channels which seem to go no deeper than the ansa lenticularis and stop short of entering the substance of the globus pallidus. This is in conformity with Alexander's findings as well as those of Addie, both of whom demonstrated a rather sharp cleavage between the blood supply of the neo-striatum and that of the pallidum, the latter in its medial portions being largely nourished by the anterior choroidal artery.

This tendency on the part of the striato-capsular arteries to avoid entering the pallidal portion of the basal ganglia and the adjacent portion of the internal capsule is

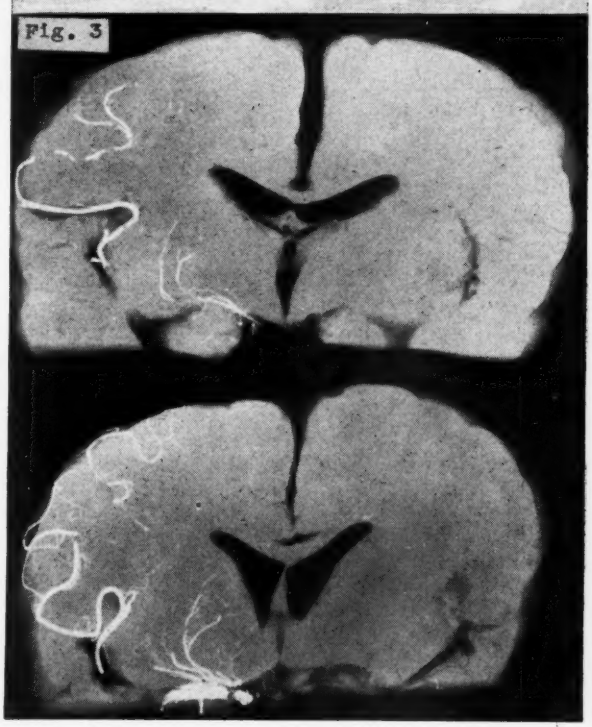

Figure 2: Simultaneous injection of perforating and cortical branches of the middle cerebral artery. No arterial anastomosis is demonstrated between the two systems across the external capsule.

Figure 3: An injection of both anterior choroidal arteries, demonstrating the many recurrent and perforating branches into the crura cerebri and the absence of prominent anastomoses with other arteries. The long recurving terminal branch on the right side is probably a communication with the posterior choroidal arteries. No spilling-over into the striato-capsular arterioles is revealed.

further demonstrated in Figure 2 (A and B). Here simultaneous injection of both the perforating arterioles and of the cortical branches of the middle cerebral artery were made. The injections further show a total absence of arteriolar anastomosis extending from the island of Reil and across the external capsule and the claustrum to the striatum and internal capsule, a cleavage which has long been recognized and particularly emphasized by Freeman.[9] While occasional vessels of arteriolar calibre do enter the corona radiata from the cortical vessels, it appears that the internal capsule, itself, is supplied entirely by a capillary bed except for the traversing arterioles to the caudate nucleus mentioned above.

Alexander has shown that only the anterior limb of the internal capsule and its very dorso-lateral portions are supplied by the perforating arterioles passing through the striatum. The knee of the internal capsule is usually nourished by short arterioles arising directly from the internal carotid.

A demonstration of the blood supply to the posterior limb of the internal capsule and of its retrolenticular portion is a far more difficult matter because it involves one of the most complicated vessels in the entire cerebral circulation, namely the anterior choroidal artery. No two-dimensional scheme can hope to plot its devious course around the midbrain and its elaborate recurrent branchings into the substance of the crus cerebri, the globus pallidus, the hippocampal gyrus, the external geniculate body and the internal capsule. Its terminal branching into the choroid plexus, which has given it a misleading name, is of least clinical importance. This very long vessel, with its important perforating branches is most predisposed to thrombosis, producing the syndrome of contralateral hemiplegia, hemianesthesia and hemianopsia, a type of ictus all too frequently overlooked by clinicians who fail to distinguish it from the garden variety of apoplexy.

With the technique applied in this study an injection of the anterior choroidal artery has not produced entirely satisfactory results because of its length and anatomical complexity. In Figure 3 both anterior choroidal arteries have been isolated from all other elements arising from the Circle of Willis and have been injected as they emerge from the stumps of the internal carotid trunks. No clear-cut arteriolar anastomosis could be shown either with the opposite companion artery or with adjacent vessels, except for a long, recurrent branch in the neighborhood of the choroid plexus on the right side, which was probably a true anastomosis with the posterior choroidal branches of the posterior cerebral artery.

The most proximal branches of the anterior choroidal artery are the sharply recurving vessels to the globus pallidus none of which reveal any supra-capillary anastomosis with the perforating arterioles of the striato-capsular group. Branches penetrating into the crura, the gyrus hippocampus and the external geniculate body do not appear to enter very deeply into any of these structures, emphasizing again the well accepted fact that the anterior choroidal arterioles are not only slender, fragile and long but are truly end arteries, all of which contributes to ready pathological conditions such as thrombosis, hemorrhage and carbon monoxide encephalopathy. Arterioles extending beyond the globus pallidus into the posterior limb of the internal capsule could not be revealed by the injection technique with the assumption that like the anterior limb, this structure contains no vessels of supra-capillary size.

Summary

An injection technique for the study of the arterial supply of the human brain at postmortem has been presented. The method permits injection of vessels down to pre-capillary diameter. The color-fast injection mass is readily hardened, it does not distort the histological picture and roentgenographs of terminal ramifications are readily obtained due to its radio-opacity.

This technique fails to demonstrate any pre-capillary anastomoses within the substance of the internal capsule. Only a few arterioles were found in the internal capsule, and these merely traversed it to find a termination in the head of the caudate nucleus.

The internal capsule is nourished by a capillary bed which is essentially a pooling of the terminations of the arterioles of the anterior perforated system of which the striato-capsular arterioles and the anterior

choroidal artery are the most important components.

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Role of Emotion in Glaucoma

C. S. SYKES, M.D.*
Galveston, Texas

Since an experience with a particular glaucoma case in the early days of my practice I have been intensely interested in the relationship of emotion to glaucoma. Every ophthalmologist encounters cases which are definitely connected with some mental upset such as grief, disappointment, fear, etc. Yet in the literature there is a surprising paucity of reports on this subject. It may well be that the oculist, having his attention focused on the diseased eye, sees that structure alone. It is my opinion that the psychic state of the patient is of extreme importance and should be considered, not to the exclusion of accepted methods of therapy and surgery in increased ocular tension, but as an adjunct to our armamentarium if we would secure the best results.

The effect of the mind on the body has long been known and recognized. As far back as the ancient Greeks it was evidenced in a statement attributed to Hippocrates.[1] "In order to cure the human body it is necessary to have a knowledge of the whole of things."

Much has been written of disease due wholly or partly to emotional factors. We are all aware of the profound influence emotion exerts on the vegetative, nervous system, such as temporary suppression of menstruation and urinary secretion, redistribution of blood supply to the pelvic organs, changes in blood pressure and respiration, sudden graying of the hair, effects on the gastro-intestinal, cardiovascular respiratory and endocrine systems. The following are examples of the eye changes resulting therefrom: protrusion of the eyes, widening of the interpalpebral aperture, lacrimation, enlargement of the pupils, spasm of the retina, weakening of accommodation and many others. Would it not be wise, therefore, to think of a patient's mentality in the practice of ophthalmology in general and glaucoma in particular? Fuchs[2] once said: "We must remember we are treating patients not eyes and must, therefore, take into consideration all the physical and mental factors which may affect his outlook upon life."

That emotion plays an important role in glaucoma has been known for over a century[3] as shown in reports by Demours in 1818, von Graefe, Donders and De Weckers.†

I wish to relate four cases which I have followed for a long time, and which illustrate the very close connection between glaucoma and the psychic state. It is not claimed that emotion is the sole cause of the disease for the eyes were not normal in the first place, but to indicate rather the role it plays as a precipitating factor.

Case Reports

Case 1: Mrs. B. P., age 32, was seen for the first time on August 12th, 1928 for a refraction. She was wearing O.U. +6.25S +1.25 Cx 90. Vision in each eye corrected was 20/25. The cornea was smaller than normal, anterior chambers somewhat shallow, tension 20 m.m. Schiotz. After solution of homatropine 2 per cent was instilled four times she was brought into the refracting room and better than 20/200 could not be obtained by any correction. During this time she mentioned that she was becoming nauseated. Digital examination revealed hard eyes and Schiotz tonometer registered a pressure of 65 m.m. O.U.

Administration of eserine 1 per cent was started immediately and instilled every fifteen minutes. In four hours the left eye had a tension of 23 but the right eye was still 65. As there was relatively little response in the right eye by the next morning, a paracentesis was done, but without success. As no improvement was noticeable after forty-eight hours an iridectomy was performed. Convalescence was uneventful. Vision of 20/40 and normal tension has been the condition

*From the Department of Opthalmology, University of Texas, School of Medicine.

†A most convincing article on this aspect is the classical paper of Ludwig Laqueur,[6] a Strasbourg ophthalmologist, who suffered from glaucoma. His portrayal of the part that fear, worry and excitement played and his own reactions open the door to the mentality of the victims of that disease. It should be compulsory reading for oculists.

of that eye since.

Later, careful interviewing brought out that after her husband was killed in a train wreck two years previously, she had had episodes of slight blurring of vision with halos around lights. I assumed that she was then going through the prodromal symptoms of an acute glaucoma. She was advised of the possibility of future trouble with the left eye but was given a hopeful prognosis if she would use immediately a 2 per cent pilocarpine solution should the symptoms reappear.

About six months later she came to my office with the complaint that the night before while attending a movie, vision in the left eye became blurred. She went home immediately and upon looking in the mirror noticed that the pupil was dilated. She instilled the pilocarpine promptly. When I saw her the pupil was constricted and tension was 18. I advised her to call me without delay if the symptoms returned.

Within a year she telephoned one evening that the eye felt "funny" again. I saw her at my office a few minutes later and found the pupil semi-dilated, tension 45 m.m. Instillation of pilocarpine 2 per cent brought the tension down to 22 in two hours. Questioning revealed that she had had a minor dispute with her sister. Since this incident I have seen her in ten similar episodes, every one of which followed some mental upset: refractory conduct on the part of her son, death of a friend, difficulties at the office, etc.

It is noteworthy that the attacks were farther apart. She asserts that she has over a period of time taken a more philosophical view of life and has not let reverses, disappointments or anger upset her. I cannot help but believe that she is right. Strange to say that while her son was away in the Pacific, during the War, she did not suffer a single attack. The fact that he came back safe and sound may explain part of it. When last seen, one year ago, tension was 20 in each eye. Left fields normal and right slightly concentrically contracted.

Here is an individual who had prodromal symptoms soon after a tragedy; first acute attacks after mydriasis and recurring episodes in the unoperated eye whenever she was mentally upset. Her knowledge of the effect of the mind on this condition and her willingness to carry out measures advised, plus encouragement and sanguine statements from her physician has enabled her to retain normal vision without operation.

Case 2: Mrs. D. F. S., age 44, was first seen May 3rd, 1931 complaining of a very painful right eye. Anterior chamber was practically non-existant, pupil fixed and dilated, steamy cornea and tension 70 mm. She was hospitalized and after unsuccessful attempts to reduce pressure an iridectomy was performed the following day. Convalescence was uneventful and final vision 20/25.

Later interrogation revealed that her husband had suffered severe financial reverses and they were in dire financial straits. She began to have evanescent spells of blurred vision shortly after she became aware of his predicament. She was dismissed with warning of possible involvement of the fellow eye and instructions as what to do should the symptoms reappear.

She went along well enough for ten years, until June 11th, 1943, when she telephoned from a distant point that she thought she was having the same trouble with the other eye. Examination revealed the same picture as first seen in the other eye. An iridectomy was done twenty-four hours later with final vision of 20/30.

She told me the trouble started soon after her husband announced that he intended to sue for divorce. Questioning brought out the fact that she had not carried out the instructions she had received should anything go wrong with the eye.

She has not been seen for five years but relatives have reported that she is doing well. Here we have one whose attacks were unquestionably provoked by mental upsets but who failed to act promptly when signs of impending crisis appeared. Operation on the fellow eye might have been avoided by the carrying out of instructions.

Case 3: Mr. R. I. C., age 39, was first seen April 5th, 1934 for a routine refraction. Nothing organically abnormal was found. He was an extremely tense individual working under such trying conditions that this tendency was aggravated.

He returned every two or three years for refraction until 1943 when he came to me

saying that he felt his vision was failing. Tension then was 30 m.m. in each eye. He was given pilocarpine with poor results. Pressure ranged from 28-35 mm. but fields were normal. He was working under great strain increased by the man power shortage of that time and his son was a combat pilot in the Pacific. While the latter situation was unavoidable, I did feel a change for the better could be made in his working conditions. That was arranged so he was given another assignment which was more to his liking and he was under a less demanding superior. His ocular tension soon dropped to 23-25 m.m. in each eye and remained that way with no changes in vision or fields until two years ago when tension was then 30 m.m. in the right eye and 39 m.m. in the left. He revealed to me that his employer was threatening to reduce his salary.

The tension stayed at that level until he received an offer of a better job in another town. The tension then dropped again to normal limits and remained so when he left in a happy and optimistic mood.

Six weeks later he returned in a badly shaken state of mind. An ophthalmologist to whom he had been referred had such an alarmist attitude that the poor fellow felt blindness imminent. Needless to say the tension was up again and higher than I had ever found it before, 39 m.m. in the right eye and 45 m.m. in the left. Offered a more hopeful prognosis he was immensely relieved and pressure fell to 22 m.m. in the right and 35 m.m. in the left. It has not been below thirty in the left since. The left fields have become slightly constricted concentrically; so he will be a candidate for surgery at an early date.

This case demonstrates what can be accomplished by having the patient avoid tense situations and the importance of the doctor's attitude.

Case 4: Mrs. H. J. A. was first seen on May 5th, 1941 at the age of 69 for a refraction. Corrected vision was O.U. 20/25. Three years later she was refracted again with similar good results. The next year, upon the death of her husband, she came to see me complaining that her vision was very blurred. Visual acuity was O.U. 20/40 and tension O.U. 39 m.m. She was placed on pilocarpine with resulting reduction in pressure to vary between 28 and 33 m.m. There was already moderate nasal shrinkage of both fields and definite vertical elongation of the blind spot. Iridencleisis on both eyes brought the tension to normal but the condition continued to deteriorate to the point now where the left eye is amaurotic and right has vision of 20/100 with the inevitable loss of that I believe.

Here we have one whose tension had been normal for years only to rise sharply when seized by uncontrollable grief with a comparatively rapid deterioration of vision in spite of operative interference and medical therapy.

Discussion

The question is how do emotional upsets bring about these changes? Schoenberg[4] concluded that the autonomic nervous system and endocrine glands effect every part of the body including the eye. He further stated that the sympathetic and parasympathetic are out of balance within glaucomatous eyes. As evidence he pointed out that:

1) Power of accommodation is diminished and there is a tendency toward widening of the pupil.

2) A solution of epinephrine hydrochloride dilates the pupil when instilled in the fornix of a glaucomatous eye which is under the influence of a miotic, whereas a normal eye is uneffected.

3) Pupils of glaucomatous eyes usually need a stronger solution for contraction (showing parasympathetic is hypotonic).

4) Ordinary doses of pilocarpine and physostigmine reduce pressure in certain glaucomatous eyes but cause no reduction in normal ones.

5) Emotional upsets, febrile diseases and sleeplessness which have a strong influence on autonomic nervous system also have a tendency to increase ocular tension.

Hess[5] believes that there exists vegetative nervous system control over ciliary body. He considers that regulatory mechanism is a reflex arc consisting of 3 parts: an afferent (proprioceptive), an efferent motor or secretory and central ganglionic, ciliary ganglion. There is second (diencephalic) arc. Irritation on any of those parts could upset the normal mechanism which is controlled by the ciliary ganglion. A third arc extends from the cortex

to the thalamus which could relay impulses such as fear and emotion to the hypothalamic organ.

Our knowledge of how the neurovegetative and endocrine influence intraocular tension is hypothetical and obscure. Medicine must advance further before the true nature of these relations can be solved. Every attempt that has been made to explain the etiology of glaucoma on the basis of one cause or another has invariably been discredited.

The question may be asked, now that we realize there is a definite relationship between emotion and glaucoma, of what practical value is it and how might we utilize that knowledge? The answer is that we know the beneficial results on the course of certain cases of glaucoma that may be obtained by having the patient as free as possible from a depressing environment. Reassurance and encouragement by the doctor is of great value in handling many glaucoma cases. An understanding of the patient's problems is an aid in guiding these unfortunates.

It has been suggested that the aid of a psychiatrist be sought in those cases where the ophthalmologist has neither interest nor time to delve deeply into their psychic background. That is one answer, but I believe the ophthalmologist can go a long way in changing the mental outlook of these people. He must be aware of the contingent condition that accompanies glaucoma and not give his entire attention to local therapy and surgery.

Realization of the inter-relationship between the mental state and the increased ocular tension should convince the ophthalmologist who contemplates an operation for glaucoma on one eye to use a miotic on the other whether the tension is normal or not. There have been many attacks brought on by worry or fear of surgery.

In conclusion I wish to emphasize the psychological care which should be used in the handling of these cases. If there is any group who should not be alarmed it is these patients. Talk of on-coming blindness is cruel and unnecessary. Most glaucoma victims do not have the capacity to take such shock without severe mental and organic damage. Glaucoma is not a disease of the eyes alone. They are sick eyes within a sick body. Every reasonable encouragement and hope should be given, for then one phase of our therapy will be successful. Of course there are some people who must be rudely frightened into returning for observation and treatment, particularly in simple glaucoma, the success of glaucoma social service in larger eye clinics shows that, but I believe they are in the minority.

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*Fatal Aplastic Anemia During Anti-Convulsant Therapy**

(REPORT OF A CASE DURING PHENURONE THERAPY AND FOLLOWING MESANTOIN THERAPY)

FRANCIS M. FORSTER, M.D. and KALMAN FRANKEL, M.D.
Philadelphia, Pennsylvania

At the 1948 meeting of the American Branch of the International League against Epilepsy, Gibbs[1] presented his results with Phenurone in the therapy of epilepsy. Phenurone, phenacetyl urea, is an unusual anti-convulsant in that it is a straight chain compound. Gibbs states it is an effective anti-convulsant in grand mal, petit mal, and in psychomotor epilepsy. Its efficacy in treating psychomotor epilepsy surpasses that of other anti-convulsants. Toxic manifestations of Phenurone therapy are exaggeration of personality defects, anorexia, feeling of weakness, headaches, insomnia, palpitation, and rash. Gibbs did not note any instances of alterations of the blood picture in his cases nor did the animal studies carried out by Abbott Laboratories reveal such alterations.

Following this report, those patients in the Epilepsy Clinic of Jefferson Hospital who had psychomotor seizures unrelieved by other medications were placed on Phenurone. One of these patients developed a fatal aplastic amenia and is herein reported.

Case Report

T.N., a white male, age 36, an electrician, was followed in the Epilepsy Clinic of Jefferson Hospital since June 12, 1947. In June 1945, at age 33, the patient had his first convulsive attack; a generalized seizure. All the grand mal attacks were nocturnal and no note of an aura was available. His wife was awakened by the seizure which consisted of a "gurgling noise," clonic movements of all four extremities, tongue biting, incontinence, return to sleep, and generalized muscle aches and fatigue on the following day. There were five generalized seizures, all nocturnal, occurring in June 1945, January 1946, October 1946, February 1947, and May 31, 1947.

In addition there were short attacks in June 1947. These lasted one to two minutes and were characterized by loss of consciousness, mumbling, clenching and unclenching of hands, and bolting his food if they occurred while eating. He had a total amnesia for these psychomotor seizures. The exact frequency of the psychomotor seizures was not known but had been at least one per day over a several week period.

The family history was negative for epilepsy and convulsive disorders in the direct and collateral lines. The patient's past history was as follows: He was the first born. Birth and delivery were normal. There were no serious illnesses and no severe head trauma. The patient had bumped his head as a child when playing but without disturbance of consciousness or sequelae.

General physical examination revealed marked varicosities of both legs and lichen planus. The neurological examination was negative.

A survey of the patient's mental status revealed a mild impairment of memory and concentration, personality change, irritability, and difficulty with the Serial 7 test.

An eight lead EEG performed on June 12, 1947 showed scattered single spikes in all leads, but more marked on the right, and with phase reversal on push-pull to right parieto-temporal region. This was interpreted as suggestive of a focal epileptic discharge.

In view of the focal EEG disturbance, mental status, and the onset of seizures late in life, the patient was admitted for study to the Neurological Service of the Jefferson Hospital on July 7, 1947. X-rays of the skull were normal. Visual fields were full on perimetric testing. The spinal fluid was under an initial pressure of 160 mm. water; there was one cell, total protein of 50 mgm. per cent, flat colloidal gold curve, and negative serology. Urinalysis was normal. Blood studies at that time revealed a red blood cell count

*From the Department of Neurology of the Jefferson Medical College, Philadelphia, Pa.

of 4,700,000 with 85 per cent hemoglobin, and a white blood cell count of 7,200 with 56 per cent neutrophiles, 30 per cent lymphocytes, 8 per cent monocytes, and 6 per cent eosinophiles. Blood Wasserman and Kahn tests were negative. The patient refused an air encephalogram and signed his release on July 14th. He was placed on Dilantin Sodium 0.3 Gms. and phenobarbital 0.1 Gms. per day and referred to the Epilepsy Clinic.

The patient appeared in the Epilepsy Clinic on September 24, 1947 having had two grand mal seizures during the day. There was no note of focal onset according to his wife who witnessed these attacks. He had continued taking Dilantin in 0.3 Gm. doses daily, but had discontinued the phenobarbital shortly after leaving the hospital because it made him drowsy. Dilantin was increased to 0.4 Gms. per day. He then complained of flatulence, but continued on this medication until October 28, 1947 when he reported having had a psychomotor seizure on October 4th. He was then placed on phenobarbital 0.08 Gm. daily in addition to 0.4 Gm. Dilantin Sodium. On November 22, 1947 the patient had a nocturnal grand mal seizure. Medication had been irregular since he felt it made him drowsy. Dilantin Sodium 0.4 Gm., Mesantoin 0.15 Gm., and phenobarbital 0.03 Gm. per day were prescribed on November 26, 1947. In January, 1948 he had three grand mal attacks within eight days while taking this medication. These attacks had an onset with head and eye deviation to left which occurred at the time of loss of consciousness. An EEG with Pentothal activation performed at this time brought out sharp spikes occurring about 6 per second during the drowsing phase and elicited from the anterior portion of the right temporal lobe.

He was again admitted to the Jefferson Hospital on February 16. Studies revealed normal x-rays of the skull; the spinal fluid was under 165 mm. water pressure, and contained 2 cells, total protein of 68 mgm. per cent, negative Wasserman test and flat colloidal gold curve. Blood studies at that time revealed a red blood cell count of 3,650,000, hemoglobin of 78 per cent, white cell count of 5,000 with 56 per cent polymorphonuclear cells, 40 per cent lymphocytes, and 4 per cent eosinophiles. EEG studies under oral seconal sedation again demonstrated the right temporal focus for the origin. Air encephalogram showed normal sized ventricles with no distortion or encroachment. On February 25 he was discharged. He returned to the Epilepsy Clinic and was placed on Dilantin Sodium 0.4 Gm. and Mesantoin 0.3 Gm. daily. He reduced the Mesantoin to 0.2 Gm. because of drowsiness and on March 9th had a psychomotor seizure. The patient complained of repeated headaches and intermittent diplopia.

Early in April he developed patches of mucositis on his palate and on the 18th he had another psychomotor seizure. Between April and June 23rd while on Dilantin 0.4 Gm. and Mesantoin 0.2 Gm. and occasionally 0.25 Gm. per day he had one psychomotor seizure. He then developed anorexia and was told to omit Mesantoin. During a seven day interval the patient had two grand mal seizures. On July 21st he was started on Phenurone therapy, receiving 1.5 Gms. per day (0.5 Gm. t.i.d.) with no other medication. When he was seen on August 4th he was irritable and complained of insomnia but he had had no attacks. Blood studies at that time showed a red blood cell count of 3,760,000, with a hemoglobin of 10 Gms., white blood cell count of 4,300 with 60 per cent polys, 37 per cent lymphocytes, and 3 per cent eosinophiles.

Phenurone, 1.5 Gms. per day, was continued. On August 18th he was in better spirits, having been free of seizures. On September 8th the patient, complaining of shortness of breath, mild swelling of the ankles, and nocturia, was referred to the Medical Clinic where it was found that he had a mitral systolic or presystolic murmur heard over the entire precordium, B.P. 130/40, and severe varicosities of both legs. The impression was mild cardiac decompensation and rheumatic heart disease and nephritis were also considered. Urea nitrogen was 19.2 mgm. per cent; urea clearance was 37 per cent, with a low urine volume; EKG was normal. The patient's next visit to the Epilepsy Clinic was on September 22nd. He had discontinued Phenurone two days previously. He complained at this time of meralopia "looks like snow on the ground," marked acuity of smell with olfactory hallucinations, persistent shortness of breath, pains in his thighs, and numbness and tingling of his hands. He was placed on Dilantin Sodium 0.3 Gm. and phenobarbital 0.1 Gm. daily and sent immed-

iately to the Medical Clinic. Four days later, on September 26th, the patient had a nocturnal grand mal seizure followed by one hour of sound sleep, awakening in a confused and disoriented state.

On admission to Jefferson Hospital the next morning, he was markedly dyspneic and had generalized cyanosis, marked engorgement of the cervical veins, rapid heart rate (132), congestion of both lung bases, and slight pitting edema of the ankles. His temperature was 98.4 degrees, B.P. 145/70, and respirations of 70 per minute. Neurological examination was normal. Blood studies revealed a red blood count of 1,200,000 with less than 49 Gms. of hemoglobin, and a white blood cell count of 1,600 with 61 per cent polys and 39 per cent lymphocytes. N.P.N. was 50 mgm per cent and CO_2 capacity 24 per cent. Bone marrow examination revealed a markedly hypoplastic marrow; there was no shift to the left nor were megokaryocytes noted. The patient was placed in an oxygen tent, was digitalized, given 2000 cc. of 5 per cent glucose in water hypodermoclysis, and a transfusion of 500 cc. of compatible blood begun. After 250 cc. of blood were administered he had a chill and the temperature rose to 104 degrees. The transfusion was stopped. Two hours later his temperature came down to 102 degrees; respirations became labored and slow, the heart rate decreased to 50 per minute. Eighteen hours after the seizure and five hours after the chill during transfusion the patient died.

Autopsy revealed marked pulmonary edema, peripheral pulmonary atelectasis, clinical anemia, hypoplasia (gross) of bone marrow, subepicardial and subendocardial petichial hemorrhages, rheumatic heart disease with chronic aortic valvulitis and minimal chronic ventral valvulitis, and sub-acute glomerulonephritis. The brain was extremely pale; the hemispheres were equal and symmetrical and the convolutions and fissures appeared normal. The meninges were clear and the vessels at the base were normal. There was no gross lesion of the temporal lobes. Coronal sections of the cerebrum revealed no gross abnormalities of the cortical gray matter, the basal ganglia or the ventricular system. A massa intermedia was present. Cross sections of the brain stem and cerebellum revealed no gross lesions. Microscopic studies of the brain are not completed at the time of reporting this case.

Discussion

Introduction of new anti-convulsant drugs in the course of the past few years has been attended by certain serious complications of therapy. Tridione was first introduced by Lennox[2] in December, 1945. Nine months later the first reports appeared of severe disturbances of red and white blood cell counts in patients taking Tridione. Mackay and Gottstein[4] reported a fatal aplastic anemia and agran ulocytosis. Non-fatal alterations in blood pictures during Tridione therapy were reported by Davidoff,[5a,5b] Davis and Lennox,[6] and Perlstein[7]. Johnson and Ayer[8] described fatal aplastic anemia in a patient receiving both Tridione and Mesantoin. Bloom and Lynch[9] recently reported a non-fatal aplastic anemia occurring during Mesantoin therapy.

Up to the present there are no reports of anemia or leukopenia developing in the course of Phenurone therapy. The instant case therefore warrants publication. It is not certain that Phenurone is the agent responsible for the blood dyscrasia. While no medications other than Phenurone were administered during the last eight weeks of therapy when the severe blood picture developed, a slight but not insignificant decrease in red blood cell and white blood cell counts occurred while on Mesantoin therapy. It is not possible to determine from this single case whether Phenurone alone or in combination with Mesantoin was responsible for the hematologic picture that evolved. However, the lesson to be drawn is that all patients receiving newer anti-convulsants, regardless of their chemical structure and of previous toxicity studies, must be carefully observed for significant alterations in the blood picture.

Conclusion

A case is described of fatal aplastic anemia in a patient receiving anti-convulsant therapy. The patient received Phenurone over a short period of time and had previously received Mesantoin.

Authors' Note: Phenurone was supplied by the Abbott Laboratories, North Chicago, Illinois.

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Developmental Mechanisms in the Pathogeny of Schizophrenia

LEWIS J. SIEGAL, M.D., LL.B.
New York, New York

The segregation of this disease as a psychosis apart from all other related types of mental derangement and assigning to it the name of "Dementia Praecox" was the work of Kraepelin. The term tends to mislead in that it connotes a disturbance or alteration of tissue, which may be either of toxic or structural origin. Besides, the term is suggestive of no dependable prognostication, as the outcome is not always a dementia but rather a degeneration and/or diminution in the interests, customs and routine of the individual.

From the interpretative viewpoint it was Bleuler who later acquainted us with the term "Schizophrenia." The latter nomenclature is based upon the concept of a splitting of the psyche (mind) coupled with a resultant power to unnaturally influence the emotions and idea association. Consequent upon this, is the outstanding dissociation of affect which proceeds to the individual's detachment from contact and actuality of existence. This introversion psychosis is rooted in the patient's personality characteristics usually noticed from early life, with inclination toward withdrawal from the society of others and with limited ability to free himself from juvenile modes of reaction.

Based upon extended clinical personal observations and upon my consideration that the schizophrenic's personality disorganization is founded upon fundamental constitutional predisposition, is predicated the logical inference that such psychic disarrangement is the patient's response to life's stresses. The immediate or precipitating motivation contributing to this mental departure may include such elements as emotionally impelling forces, toxemias, head injuries, exhaustion, sex, age, environment, biological transitional periods, etc. Upon the gravity of the precipitating factor as well as the quality of constitutional deficiency of the individual will become established the ability to survive at the original level, or, to make adjustments on planes which will descend in the scale progressively until the point of frank psychosis becomes evident.

These patients will release incongruous emotional reactions and mental concepts which cannot be foreseen, constantly present unpredictable and grotesque symptomatology and exhibit a defective relationship between them.

Biologically, in the process of orderly and gradual development, consciousness of the self comes into being after the period of immaturity has given way to the state of cognition and it is at the latter stage that the psyche assumes evolutionary modification of function and gains first knowledge of that which is visible or tangible to the power of understanding.

As between the mental impressions of the

immature and those of the fully grown individual it is significant that they perceive their surroundings dissimilarly. Similarly, clinically observed, mentally disorganized subjects—because they have turned back emotional craving activating human behavior to an early fixation at a narcissistic level—become aware of their surroundings in a more opaque and unmodified manner. It is here that the breaking up of one's individual discriminative qualities takes place with what amounts to a reversion to a period when the control over functions of the organism in respect of its relationship to the external world had not yet existed, or, had just come into being. It is here that the requirement of an instantaneous release reaction is not grounded upon deliberated discriminative integrity such as is ordinarily the result of pre-determined and judicious delay of emotional response—a period during which latency crystallization of reasoning ordinarily takes place. An immediate primitive instinctual response therefore points to an objective reaction of a feeble ego (self) which has not mastered the ability to control conscious perception and acts. Consequently, in its essence, detachment of the self from the object world portrays an unfolding phenomenon characterized by gradual transformation to simple release response in contradistinction to definite bodily reactive functions attendant upon discriminative power. Accordingly, the competency to foresee precarious situations and to reason out the manner in which to attack or to obviate them, is an act expressive of an individual's ability to control conscious perception in conformity with the object world.

The stages of schizophrenic alterations in bodily functions will be developed under the following components:

A) Definite retrogradation of the self with degeneration of the psychotic's ability to discriminate in respect of that which he had gained evolutionally. These changes may be exemplified by fixations concerning cessation of world actuality, altered percepts to bodily stimuli, diminished perceptive capacity for one's identity, mental conceptions of grandiosity, reversionary intellectual function, deteriorative verbal expression and manneristic speech and action.

B) Other altered activities in which the patient may indulge in an effort at self reclamation. Under these we recognize the varied disorders of perception (illusions, hallucinations), disturbances of thought content (delusions), as well as volitional disturbances. The latter may be acted out by the patient either as gradations of decreased activity or by a predominance of varying degrees of psychomotor hyperactivity.

A) Retrogradation of the self is representative of the schizophrenic's total relinquishment of object situations or ideas and as such is a state of throw back to an earlier period, with predominance in self-adoration. Such dynamic expansion of the love of self may be reflected in the patient's intensity toward some specific bodily organ or he may direct it to his complete corporeal entity. This expansive expression may frequently become perceptible to the psychotic in the form of a melancholic mental state, in which latter condition the withdrawal of his ideas and feelings from other persons or situations will enhance the mental charge toward his own body or its parts and give rise to the hypochondriacal phenomenon.

When this dynamic expression of the original expansive impulse is held in check by a superior drive vigorous enough to turn aside these hypochondriacal delusional trends, the resultant perception engendered in the psychotic is evinced by a feeling of enstrangement either of his entire body, of bodily parts, or of particular organs. It is to be observed therefore, that augmentation or diminution of corporeal sensations inevitably alter the schizophrenic's mental conception of his body and imparts to him the feeling of strangeness.

The consciousness of a lessened sense of one's own identity which may even range to total unfamiliarity with one's self, portrays a response of the self to the awareness of the growth of one's dynamic expression of self-love. Such pathological misconception is indicative of the turning back of the psychotic's libido to an earlier fixation with attendant transfer of his emotional drive to an inappropriate idea, albeit it appear logical in his unconscious. Oft times this earlier fixation, rather than being turned aside, thrives and gives rise to the sensation of an agreeable and imposing pomposity. The latter emotional charge devoted to objects external to the self comingled with those of the self,

results in the psychotic's feeling of exaltation, grandiosity and enchantment. On close observation, it will be noted that such patient refuses to accept as real his inability to possess these primitive perceptions of unlimited power and maintains the latter either through narcissistic continuance of its possession or, by re-acquirement of its control, with the result that a turning back to an earlier fixation amounts to the fundamental infantile misconception of instantaneous wish fulfilment. Upon this is superimposed a growth in the psychotic's conceit and high value of himself. These frequent manifestations of psychotic oversupply of disordered perceptions of personal grandeur and ideas of exaltation, serve as a response reaction in an effort to assuage the anguish of a narcissistic trauma suffered during adult existence.

In addition to his renewed disposition toward the love of himself and feeling of the all powerful, the psychotic may also be deluded with the notion that he is indulging in sexuality with his own person. The latter phantasy is not identifiable with the primitive order of development before the commencement of perception toward external things, but applies to reactivated or secondary regressed narcissism wherein external things have once more become supplanted by his ego. Such pretentiousness is a phase of the enchanting and spiritualistic existence which fantastically is apt to occur in secondary or narcissistic return to an early inferior level of fixation.

Not expressed in consonance with our approbative rules of correct reasoning, the schizophrenic's thoughts are inseparably associated with crude fantastic conceptions and as such, they fall back to the pre-reasoning stage. While his mode of thinking is correspondingly more forceful and more specific than our type of reasoning according to standard principles, the psychotic is nevertheless unable to put it to use for the mental perception of the facts of life's actualities. This failure necessarily impoverishes his adaptability and readiness for events or future deeds. Such form of reasoning is equally inadequate in dealing with outward facts in respect of events which are to come, the inability being due to remoteness from logic and its comparative unsystematic structure fraught with inconsistencies, is governed by impetuosity and laden with craved-for and dread-inspiring delusions. The immature obscure thinking of the psychotic is portrayed by unconsciously substituted objects expressive of his libidinal thoughts guided by primitive mental activity and is a mode of mental perversion which appears where regressed primitive ego peculiarities have once more come into existence.

In contrast with the non-psychotic who excludes thoughts from the consciousness by repression, the psychotic's symbolic thinking becomes conscious without his becoming emotionally displaced by these mental processes or by their results. The symbolic thinking of the schizophrenic is not just a mode of mental perversion but is his realistic primitive reasoning and perception of the symbols with introduction of material related to them. While this quality of intellectualization is equally prevalent in individuals other than psychotic, it only continues to exist as such while excluded from the consciousness by repression.

Awareness of a concept emblematic of an object which stands for something beside itself may exist in a non-psychotic, to cover up some offensive repressed mental pattern. Here the object emblem employed is consciously recognized while the representative figurative idea is not. In the regressed archaic thinking of the schizophrenic, understanding of primary thoughts where original objects represented sources of satisfaction or the coming of danger, continues indiscriminately, e.g. departure meaning death. Hence, to the psychotic, primitive object stimuli continue to elicit the identical emotional answers and are perceived by him in an undiscerning manner because he does not recognize the difference between absolute sameness and general resemblance.

In the psychoses coming on at about the pubertal period, deprivation of interest directed toward external objects or things is definitely apparent and the submissive character of the protective process of reversion to an earlier fixation level is clearly obvious. Here the individual personality puts up no protective barrier when confronted by turmoil, but characteristically withdraws to a former period of life taking shelter in different and perhaps antiquated adjustments, reaching to the level of primitive submis-

siveness—even possibly down to the prenatal stage—evincing no more than just passive involuntary functioning.

In the transformation of one idea into another, a substituted mental impression aware to the senses may be employed by the non-psychotic individual in order to obscure an unwanted and unconscious hidden mental conception. In this situation the substitutive expression thereof is conscious, while the character of the undesirable hidden mental concept is not. However, in the archaic mental activity as found in schizophrenic regression, the primary notions of objects which gave to the psychotic original percepts of pleasure or danger, continue without his awareness of their dissimilarity. To such patient the primitive object stimuli persist in bringing forth these unchanged emotional reactions which are perceived by him in an undistinguished manner because of inability to discriminate between likeness and sameness.

Inattentiveness to object situations and unresponsiveness to external stimuli can be well observed in the deprivation of the *hebephrenic*. His ego will put up no resistance when confronted by strife but will regress to time gone by and find shelter in different (even archaic) modifications, with adjustments ranging down through the simplest childhood stages to the earliest period of fetal involuntary function.

Repressed individuals often remain comparatively stable during the interval between infantile sexuality and onset of pubescence, but will later abandon novel ideas or accomplishments because of inability to handle the unconscious emotional stimulations innately activated at attainment of corporeal sexual maturity. Here such patients may be observed in bouts of devastating violence which reactions they had priorly kept in check by a mild passive submission. In their self-adoration and retrogradation they may appear to arouse sorrow and pity while underneath they harbor grave antagonism concerning persons or things external to themselves. This division between emotion and thought formation has for its purpose the casting aside of determined melancholy dreaded conscious feelings. With the breaking up of the protecting ego with insensibility to the surrounding objectivity this purpose is transformed into an obscurity by detachment from individuality, with a resultant purposeless and vacuous aspect of countenance. Aimless activities and grotesque postures in addition to being representative of remnants of destroyed object relationships, more particularly portray an unsound effort which lacks the ability to restore such contact. It is an ineffectual struggle for reclamation to a former state.

The mechanical submissive compliance of the *catatonic* with his meaningless repetition of words and gestures is indicative of his childlike method of imitative representation in an effort to associate with other individuals by mimicking and repeating the gestures which he sees about him. The latter, derived from the catatonic's employment of primitive recognition, appear as manneristic bodily movements and facial expressions indicative of amusement. They do not merely duplicate what he has already seen, but equally foresee, by reason of these immature performances, the longed-for deportment of other individuals.

The inflexibility as a mechanism of the catatonic is demonstrative of a struggle between the incitement to act and the protective effort against it. This is in actuality a cataclonia—a characteristically persistent and repetitious cycle of stimulative and suppressive activity. The latter are expressions of self-centeredness or converge around the conflict to recover external relationships. As this reversion progresses to a lower level, desires to recover external relationships dwindle while they increase in intent to refute disagreeable sensations. These discordant cataclonic releases serve to ameliorate the psychic stress and are likely to give way to a total and sustained bodily immobility.

B) As efforts at self-reclamation, manifold grotesque notions are met with as the psychosis progresses. The patient may believe that it has fallen to his lot to bring order out of world chaos through Divine Command, that he is the savior of the universe, or that the world is to take on a new existence. In some patients, I have observed that these emotional states will stimulate them to rapture, while in others these drives engender marked terror. At this stage, these are representative sense presentations reacting in the psychotic in his effort to reclaim that which he was deprived of through morbific object

perception, the terrorizing or rapturous quality of these drives portraying the despairing or sanguine reaction to this undertaking. Restorations are often felt submissively by the patient as a primitive object perception. Indulgence in piety (which beliefs predominate in this mode of reclamation) accompanied by demonstrable chatter and symbolic of the father's reigning supremacy, affords to the patient an ideal chance to reveal his contradictory clashes in respect of his repressed father concepts. It is this mechanism that is indicative of the psychotic's confusion, with the demonstrable clashes evidencing his effort to put up a defense against the abnormal or negative Oedipus complex.

Due to the biological withdrawal of external sense perceptions during sleep, thoughts which are perceived without an object and called dreams, are but hallucinatory or substitutive perceptions. The morbid retraction by the schizophrenic of mental stimuli surrounding an idea or object gives way to the identical hallucinatory consequence. During our normal dreams we experience the imaginary production of more satisfying perceptions in place of disagreeable realities. The schizophrenic dodges his struggle between opposing desires by the repudiation of reality, submerging distressing ideas into the unconscious by returning to the archaic condition of falsely perceived wish achievement in which he existed before he was able to determine that which exists in actuality. In this manner the schizophrenic replaces, by perceptions having no relation to reality or to actual external stimuli, his mental concept of recent objects or stimuli, where the reception of fresh genuine mental impressions thereof has been impeded. The false sensory perceptions commonly experienced by this type of psychotic have demonstrated to me that in the majority of instances they are distressing and terrifying to the patient and do not reflect ordinarily attained longings. These mental wanderings (not based upon reality or upon external stimuli) are not merely representative of the flight of the conscious self-identified personality, but in like manner characterize unattained flight—a perverted reappearance of averted mental incitation to unpremeditated acts in the pattern of imputation of one's own notions to others. The hallucination which typifies menace or castigations directed at the inner subconscious censor which the conscious self-identified personality had attempted to oppose, is expressive of symptoms of hearing, mental in origin, emanating from the inner subconscious censor. This symptom accounts for the verbal auditory hallucinations experienced by this class of psychotic individuals. Such distorted perception is not merely representative of the resultant of a renunciation of that which exists in actuality, but applies equally to a rejection of the unconscious dominated by its instinctive impulsions, or, of the inner subconscious censor.

Delusional mental misconceptions are representative of unsound mental perceptions of actuality founded upon distorted cognition and may be displayed as an awareness through the senses or as a delusional interpretation applicable to life's ordinary incidents. These reflect an effort at replacement of the missed elements of actually existing objects or experiences and are often laden with segments of renounced actuality, of resisted impulsions and of ideas or motives attributed to others, exacted by the inner subconscious censor.

The delusional individual is especially prone to a distorted consciousness of the content of awareness of other persons and employs this tendency in order to assign a plausible explanation for his bizarre and manifold ideas or motives imputed to others.

Oft times, mechanical contrivances (symbolic of a replica of the psychotic's own body or organs) torment and impel the patient, by way of his inner subconscious censor, to impute to others such motives or ideas as that he is being watched, censured, supervised and penalized. The latter outward rebukes commonly occur as vocal utterances, which, if they denote to the patient the percept of being castigated, lessen his conscious sense of culpability and vindicate his antagonism. The constant impression that he is being maltreated by others serves as an extension of his justification for the contradictory conscious awareness of his culpability. It is at this point that the primitive self-admiring character of this type of psychotic manifests itself, by an exaggeration of his righteousness and guilelessness which stems from this retrogradation to primitivity.

These fanciful mental excursions and their

quality, are obvious reflections of the *paranoid* schizophrenic's transitional attempts to extricate himself from this condition of primitivity in an effort to establish a relationship with the world of reality and to reach out for his former place in mankind's association. These fragmentary mental stimuli may take on numerous peculiarities, some of which will turn the paranoid schizophrenic's attention to the archaic sphere of desire for stimulation of mucous surfaces. The desires may take on such apparent pleasurable sensations as evacuation of intestinal contents without orderly utilization of the ward lavatory, infantile attraction to excreta, emptying of the bladder, abnormal intake of food, total abstinence of food necessitating tube feeding, archaic modes of mutual association, or insatiable desire to assimilate objects by oral introjection, characteristic of these patients' primitive effort at identification with such objects—a noteworthy remnant of infantile cannibalistic manifestations. It is the consummation of these abnormal desires by this class of patient that poses the constant and difficult task to the personnel charged with the problem of keeping these patients and maintaining our wards which house them, in a fragrant condition.

Precisely as the child attains imitative capacity for association with others by copying their expressive motions and attitudes through repetition of words, actions and unconscious submissiveness, so does the stuporous or *catatonic* type of schizophrenic employ the archaic instinctive drive of early assimilation, in a regressive struggle to recover his association with lost object relationships. The grotesque simulated movements and acts do not merely copy what the catatonic has observed at a former time, but equally reflect by his expectation through mysterious bodily attitudes, the aspired deportment of others in time to come.

Stupor and muscular tension is indicative of the incompatible drive between action and no action. This denotes the catatonic's object sense which is primitive or points to his attempt to repossess interest in persons and things external to the self. These peculiarities are frequently demonstrative of a consciousness of illness or of a repudiation of a disagreeable sensation. In the more deteriorated subject, careful clinical observation will disclose that these unrelated muscular activities are expressions of widespread bodily hypertonus—catalepsy. Such bodily performances carried out by the catatonic in his effort to reduce the severity of his mental strain, may assume such proportions that no regard for his safety becomes material to him—even to the point of carrying out self-inflicted mutilating operations upon himself.

The precipitate phases of mental disease are not materially dissimilar to the component etiological elements of the neuroses. They are progressive growths of unconscious stress distinctive of the principle that the schizophrenic psychoses oftentimes have their inception during pubescence or, in situations which excite subdued immature erotic impulses particularly homoeroticism and infantile pleasure through intestinal evacuation, or, situations which give reason for immature anxieties and sensations of guilt.

The influence of interplay is exerted between these unexpectedly-occurring elements and the innate organization of the individual. Illustrative of this are the psychoses which frequently have their inception at a time of pressing exigency during which period some circumstance deranges the individual's mental poise contemporaneous with approaching inadequacy of the protective forces of his conscious personality.

These responses therefore, are awakenings and increased attachments to primitive erotic phantasies and, characteristically, unnatural primitive erotic enjoyment oftentimes colors the schizophrenic's past record of events.

Conclusion

It is of particular importance, especially in our clinical consideration of young schizophrenic patients to properly evaluate diagnostically, the incipient manifestations of their psychodynamics anticipatory to the long range view of adaptability to choice of therapeutic formulation.

To date, reliance upon laboratory findings has afforded us no conclusive diagnostic criteria which could be employed to assist in the differential establishment of express schizophrenic stages from other mental diseases.

The major part of the psychiatric examination should achieve a comprehensive personality study of the individual patient. This

particular affords the disclosure of many of the symptom complexes upon the existence of which must be determined the quality of the mental disease dealt with. Only through an understanding of the nature and meaning of such personality manifestations are we able to recognize and distinguish alterations from the accepted normal and to allocate proper interpretative values to said behavior anticipatory to a determination of the criterion of therapeutic adaptation.

From my studied divers observations carried out in hospital wards, out-patient clinics, as well as non-institutional patients, all considered in their respective settings, I am amply persuaded that it is of the utmost importance, from the standpoint of therapeusis, to carefully scrutinize and evaluate all precipitate presumptive motivating factors contributive to this psychotic entity. Appropriate recognition and appraisal of these elements therefore—based upon the premise that an organism cannot perform in a degree above its innate faculty—should be aimed at in an endeavor to arrive at the pathognomonic determinants, with an outlook toward therapeutically accomplishing the patient's adjustment level on a plane higher than could have been attained otherwise.

Toward the objective of elucidation of the developmental psychodynamics and their significance in the schizophrenic psychoses has the present discourse been directed.

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Neurological Clinical Pathological Conference

OF THE CINCINNATI GENERAL HOSPITAL

CHARLES D. ARING, M.D., Editor

CASE NO. 217552

Presentation

A 19-year-old white boy was brought to the Receiving Ward on September 15, 1946, in coma. Information from the patient's mother indicated that he had been his usual self until the morning of September 11, when he failed to arise at his customary time. When an attempt was made to arouse the patient, he was unable to speak. His left extremities were weak and the left side of his face sagged. A physician summoned a few hours later witnessed a right-sided convulsion which was followed by loss of consciousness. Subsequently the patient improved to the extent that he recognized his mother and took fluids by mouth but was never able to speak. The day before admission he became progressively less responsive and developed a fever of 103 degrees F.

The only information which could be gleaned from a review of the past history was that the patient had never developed properly and was mentally retarded. He had never complained of headaches or visual disturbances; however, it was noted that he was unable to move his eyes above the horizontal plane without elevating his head. He had had occasional fainting episodes the month previous to the development of the present illness.

At the time of admission to the Receiving Ward the patient was in profound coma. He appeared to be poorly developed; the skin was smooth and unusually soft; the hair was extremely sparse over his entire body; the head appeared small. Respirations were rapid and shallow, and there was dullness to percussion over the left side of the thorax associated with increased breath sounds and occasional wet rales over the same area. The heart was of normal size, but the sounds were distant and indistinct; the rate was rapid and the rhythm regular.

The pupils were small and fixed to light. There was an internal strabismus thought to be bilateral but more marked on the left, a moderate ptosis of the left lid, and an occasional burst of rotatory nystagmus. Fundiscopic examination revealed loss of the optic cups, slight congestion of the retinal veins and a few small flame-shaped hemorrhages. There was a central type of facial weakness on the left and flaccidity of the left extremities. Random movements of the right extremities and on one occasion slight movement of the left upper extremity were observed. There was no response to corneal stimulation, pin prick or pressure over the supraorbital nerves. The deep reflexes were slightly more active on the right; the knee and ankle jerks were hyperactive; ankle clonus was elicited bilaterally; no response to plantar stimulation was obtained on the left whereas dorsiflexion was present on the right. The abdominal skin reflexes were absent.

The patient was immediately prepared for operation and taken directly to the operating room. When placed on the operating table, he ceased to breathe and the pulse rate increased to 240. Bilateral occipital trepanation was attempted, the patient being carried with the aid of artificial respiration. Ventricular puncture revealed a small amount of clear fluid under increased pressure, while the brain beneath the right burr hole was under tension. The heart had ceased to beat before the left burr hole was completed, 5½ hours after admission to the receiving ward.

At the time of admission the temperature was 105.7 degrees F., the respiratory rate 40, the heart rate 140 and the blood pressure 110/0.

Laboratory Data: Roentgen examination of the chest revealed the lung fields to be clear except for an area of increased density posterior to the heart on the left while that of the skull revealed the cranium to be small, the posterior clinoids to be thin and poorly mineralized and the sella turcica to be of normal size.

No blood studies were recorded. The protein determination on clear ventricular fluid was 365 mg. per cent.

Differential Diagnosis

Dr. Howard D. McIntyre: One must comment that more historical information is lacking than is supplied in the protocal of this case. How can we break down the data that we do have in order to arrive at a diagnosis?

A nineteen-year-old white male, awakens with aphasia which came on during the night and which was accompanied by weakness of the left face and limbs. We do not know whether the patient was right or left handed. This information would indicate that the patient had a stroke during the night.

Another pertinent fact is the right-sided convulsion observed by a physician and the coma which supervened. Following this convulsion the patient regained consciousness but remained aphasic. As we do not know the patient's handedness we do not know as yet whether the vascular basis for his symptoms was on one or both sides of the brain. It has been said that a generalized convulsion may be rendered Jacksonian-like by a hemiparesis, the paretic limbs not responding to the cerebral discharge.

Positive findings on admission to the hospital were, (1) beginning choked disc, (2) aphasia, (3) weakness left face and limbs, (4) small, fixed pupils, (5) internal strabismus, (6) ptosis left eyelid, (7) nystagmus, (8) bilateral pyramidal tract signs, (9) coma with high fever and tachycardia. These signs point to a lesion high in the brain stem at the quadrigeminal plate. Obviously at this stage of the illness tumor was suspected as burr holes were planned.

Is the past history contributory to a diagnosis? Gleaned from it were, arrest of cerebral development with microcephalus, paresis of upward gaze of unknown duration, suggesting midbrain localization; fainting spells, which are not described and which began a month before onset. The fainting could represent convulsive phenomena. In regard to these attacks I am reminded of the rare ball-valve action of tumors in the third ventricle. Soft skin and sparse hair suggest disorder of the pituitary gland or of the hypothalamus.

What is contributed to a diagnosis by the laboratory? Roentgen studies of the skull showed microcephalus and thinning of the posterior clinoids. The latter might indicate either demineralization or pressure. In view of other factors I would be inclined to interpret it as a sign of increased intracranial pressure. The sella is of normal size which would exclude pituitary adenoma. The absence of suprasellar calcification would be against craniopharyngioma where calcification occurs in 80 per cent of cases. Roentgen plates of the chest disclosed a density posterior to the heart on the left side. This suggests the possibility of a tumor in the chest with metastasis to the brain. We will return to the discussion of this problem later.

What information can be gained from the exploratory right burr hole? There was increased intraventricular pressure and no internal hydrocephalus or more fluid should have been obtained. The needle apparently was not in a cyst since the fluid was clear—cysts contain yellow fluid. The total protein of the ventricular fluid was quite high (365 mg.). All the information obtained from the ventricular fluid points more to a diagnosis of tumor than to any other process.

The small ventricle without the hydrocephalus is against a third ventricle tumor, pinealoma or other tumors of the quadrigeminal plate. The increased protein in the ventricular fluid excludes tumors below the tentorium and this information at the least restricts the problem to a consideration of supratentorial disease.

If protein determinations were available from both lateral ventricles our problem would be simplified. The principles underlying the interpretation of increased protein content of the ventricular fluid are as follows. A normal protein content in ventricular fluid with increased protein in the lumbar sac usually points to subtentorial tumor. In this condition the spinal fluid protein is increased due to block and possibly due to leakage of protein from blood vessels in and about the tumor. In subtentorial tumors the ventricular protein is usually normal. Increased protein in both lateral ventricles indicates third ventricle tumor or blocking of the aqueduct of Sylvius for any cause. The result is a non - communicating internal hydrocephalus. Tumors of the corpus callosum frequently produce increased protein in both lateral ventricles.

Although fluid from both ventricles is not available in this case, ventricular pro-

tein values of over 300 mg. do occur in third ventricle tumors, but the average protein content is from 50 to 150 mg. When the protein is increased in one lateral ventricle the tumor is on the side of the occluded ventricle, the occlusion occurring at the foramen of Monro. In such tumors the protein content of fluid from the opposite ventricle is either normal or only slightly increased. Protein values over 300 mg. in the ventricular fluid are more common in unilateral tumors occluding a foramen of Monro. Unfortunately we are unable to apply these principles with any accuracy in this case because of the fact that only one ventricle was tapped.

From the inadequate information available in this case what are the diagnoses to be considered? Certainly tumor was entertained strongly by those who examined this patient. Consideration of any other diagnosis than tumor seems speculative because of the paucity of evidence.

My diagnostic evaluation runs somewhat as follows: (1) tumor of the quadrigeminal plate: (a) pinealoma, (b) teratoma, (c) dermoid, (d) glioma, (e) metastatic tumor. Against this diagnosis is the fact that internal hydrocephalus was not demonstrated via the right burr opening. (2) Tumors of the third ventricle: (a) ependymoma, (b) subependymal glioma, (c) metastatic. Again we meet the objection that no internal hydrocephalus was found when the right ventricle was punctured. (3) Tumors in the region of the chiasm: (a) pituitary adenoma which is ruled out by the normal sella, and (b) craniopharyngioma which is unlikely because of the lack of suprasellar calcification. Tumors about the chiasm occasionally result in an increase in protein in both lateral ventricles. (4) Tumor of the corpus callosum. (5) Tumor invading the right hemisphere (perhaps metastatic from the lung) with compression of the foramen of Monro. In favor of this diagnosis is the small ventricle on the right together with marked increase in protein content of the fluid.

One might well ask the questions, "What bearing does the congenital arrest of brain development, the microcephalus, the abnormalities of the skin and hair have on the diagnosis of the terminal event in this case?" What are the modes of death in congenital arrest of the brain? What tumors or anomalies occur in congenital arrest of development which may cause such a death as this? These questions expose the paucity of first-rate monographs in English on arrested cerebral development. Text-books usually concern themselves with hydrocephalus, spina bifida occulta, cranium bifida, and cerebral diplegia; that large group of borderline cases of arrested cerebral development has not received the attention it deserves.

Dr. Charles D. Aring: My own reasoning was somewhat as Dr. McIntyre's. I hadn't heard about the patient before I read the protocol. The patient was an immature lad who had never been normal physically or mentally, and since the onset was seemingly apoplectic, I thought of an angioblastic type of lesion perhaps somewhat more wide-spread than Dr. McIntyre indicated. It would take a bit of circumlocution to delineate why the erupted blood therefrom did not reach the ventricle, but I believe such a picture could be drawn. This case brings to mind another,* involving a young woman aged 30 who had had neurological disorder practically all of her life and who suddenly suffered a stroke. A cavernous angioma involved practically the entire right hemisphere, as well as the midbrain and upper one-third of the right pons. In this case the cerebrospinal fluid was bloody.

Pathological Discussion

Dr. Ralph Johansman: The body was that of a moderately well-developed, fairly well nourished young white male; it measured 150 cm. in length. The pubic hair was sparse; the testes were small but were in normal position in the scrotal sac. The heart was of average size and weighed 275 grams. There were no deformities of the heart valves and there were no septal defects. The ostia and the main branches of the coronary arteries were normal. Several of the small branches of the circumflex branch of the left coronary artery were narrowed almost to the point of occlusion. The myocardium in the distribution of these stenotic vessels was increased in density and focally softened. A small mural thrombus was attached to the left ventricular mural endocardium overlying the myocardial lesion.

*Neurological Clinical Pathological Conference of the Cincinnati General Hospital. Case No. 44-2976, *Dis. Nerv. Syst.*, 6:119-124, 1945.

The lungs, which were increased in weight, were moderately edematous and congested. The bronchi were acutely inflamed, and ill-defined areas of pneumonic consolidation were scattered throughout the right lung. There was acute passive congestion of the abdominal viscera.

Microscopic sections showed a localized lesion in the myocardium, extending from the epicardium to the endocardium. This lesion consisted of miliary and moderately large, confluent zones of degenerated and necrotic muscle cells. In many areas muscle cells were absent but the stroma was intact. A lymphocytic reaction was present throughout the lesion, but this varied in intensity in different portions. The thrombus which was attached to the mural endocardium was undergoing organization. There was slight fibrosis but no evidence of hemorrhage or blood pigment deposition in this lesion. The histologic picture essentially is that of a subacute myocarditis and not that of an infarct. However, vascular stenosis undoubtedly played a role in the development of the lesion. There was marked localized intimal connective tissue proliferation and fibrosis in the stenotic segments of the coronary arteries and this also appeared to be the result of an inflammatory process. The past history unfortunately inadequate, offers no clue as to a possible etiologic factor.

Sections of the lungs showed congestion, edema, acute bronchitis and lobular pneumonia. Aspirated foreign material, probably vomitus, was present in some of the bronchi and the mucosa of these bronchi was necrotic and partially digested.

The seminiferous tubules of the testes were lined by normal appearing sertoli cells but there was no evidence of spermatogenesis. The interstitial cells were normal in appearance and distribution. The pituitary gland was of average size. In one section studied there was marked increase in the numbers of eosinophilic cells in the anterior lobe and this seemed to be at the expense of the other cells, particularly the chromophobes. However, the ratio of the various cells throughout the entire lobe was not established and conclusions cannot be drawn. The adrenal glands were normal. The thyroid gland was not examined. These findings when considered with the clinical evidence of mental retardation and slight retardation of somatic growth suggest that the hypogonadism might be secondary to anterior lobe dysfunction..

Dr. Murton Shaver: The autopsy had been performed five hours post-mortem. The brain, which weighed 1175 grams, showed generalized flattening of the gyri over both hemispheres with narrowing of the sulci and herniation of the right hemisphere particularly the frontal regions to the left under the falx. There was a marked cerebellar pressure cone and a small area of tissue destruction on the left lateral portion of the brain stem suggesting a Kernohan's notch. There was no definite uncal herniation at the time the brain was examined after formalin fixation. Basal blood vessels, leptomeninges, and dura were not in any way remarkable on gross inspection.

Coronal sections made through the cerebrum demonstrated marked swelling in the right hemisphere, particularly in the frontal regions where the tissue was soft and poorly fixed. There was midline shift to the left, a displacement of approximately 1 to 1.3 cm. The softened area extended from the mid-frontal region to the posterior parietal region and extended down to the basal ganglia which contained small areas of hemorrhage particularly in the region of the thalamus. Similar areas of hemorrhage with a very small area of softening were noted in the left thalamus.

The left occipital lobe showed hemorrhagic discoloration of the cortex in many areas and appeared moderately swollen and poorly fixed. The tissue was soft but not as soft as that noted in the right hemisphere and in the basal ganglia bilaterally.

Sections through the right temporal and frontal lobes in the area of softening showed early histiocytic proliferation in the tissue underlying the pia-arachnoid in some areas. No inflammatory infiltration was noted in the region of the leptomeninges although a few red cells were found in the sub-arachnoid space. The cortical cytoarchitecture was disrupted and only a few ischemic and hyperchromatic nerve cells remained. Many of these showed satellitosis and neuronophagia. No perivascular hemorrhages were observed. Many of the arteries in these sections showed complete occlusion by thrombus material and a few of these vessels showed acute arteritis

with polymorphonuclear infiltration of the vessel wall, and in some cases, this had extended into the surrounding tissue for a short distance. A few aggregations of polymorphonuclear cells were found scattered in the tissue elsewhere. No definite organisms could be identified with bacterial stains in these areas.

Sections of the stem of the middle cerebral artery demonstrated the lumen to be occluded by grayish material resembling thrombus which was slightly adherent. On microscopic section, thrombus material was identified in some sections.

One section through the temporal lobe more posteriorly demonstrated a few areas of old rarefaction of the white matter in which the myelin had been completely removed and the area of destruction surrounded by histiocytes and a margin of early gliosis. Throughout these sections, there was congestion of the venules but hemorrhages were not encountered.

Sections through the right thalamus demonstrated early softening with demylination in the region of lateral wall of the third ventricle while nerve cells showed ischemic changes and were hyperchromatic, showing acute and profound changes with loss of nuclear structure. The vena terminalis was completely thrombosed as were many of the smaller veins scattered through the basal ganglia. There was no evidence of gliosis in this tissue either cellular or fibrillary and there was no evidence of histiocytic proliferation. Sections through the posterior portion of the third ventricle at the level of the colliculi showed thrombosis of both veins of Galen with numerous hemorrhages into the colliculi and thrombosis of the pontine and basilar veins. Most of the hemorrhages were perivascular in situation, some of them being rather extensive. The nerve cells showed ischemic and acute and profound changes as described in more anterior regions. Here there was more marked swelling of the interstitial substance and the general appearance was that of an early hemorrhagic infarction involving this area. Sections more posteriorly through the colliculi and aqueduct of Sylvius demonstrated more extensive hemorrhages in the upper brain stem with patchy areas of rarefaction and edema in the white matter at this site.

Sections through the left occipital lobe demonstrated recent cortical perivascular hemorrhages which were largely situated around the small venules and were associated with the presence of thrombi in some of the smaller veins. There was no evidence of inflammatory infiltration in any of these sections, either in the vessel walls or in the cerebral tissue. The nerve cells generally showed swelling with chromatolysis but there was no evidence of proliferation of the endothelial cells and no gliosis was observed. The white matter generally in this region showed early rarefaction with swelling and vacuolization.

We are dealing with multiple emboli. Whether or not the emboli were infected cannot be determined from the histiologic study alone, but there are areas suggestive of early infection as described in some of the arterial walls and a few places in the cerebral tissue. The areas of infarction are obviously of varying age, one small area in the right temporal lobe being old, while the large area of ischemic infarction described in the distribution of the right middle cerebral artery showed early histiocytic proliferation which would probably date from the patient's earliest described complaint four days prior to admission to the hospital. More recent areas of infarction are apparent in the basal ganglia bilaterally while hemorrhagic infarction is present in the left occipital lobe and in the upper brain stem giving rise to the symptoms and physical findings described shortly prior to the patient's demise. As you have already gleaned from Dr. Johansman's description, the source of the emboli is most likely the mural thrombus in the heart.

Clinical Diagnosis

Supra-sellar cyst; left lower lobe pneumonia.

Dr. McIntyre's Diagnosis

Cerebral tumor, right hemisphere.

Pathological Diagnosis

Focal subacute myocarditis with organizing mural thrombosis.

Pulmonary congestion and edema.

Acute passive congestion of the abdominal viscera.

Acute bronchitis and lobular pneumonia.

Hypogonadism, possibly secondary to dysfunction of the pituitary gland.

Embolism of right middle cerebral artery and branches of the left middle cerebral artery.

Subsequent infarction and swelling of the right cerebral hemisphere with displacement of the brain stem, and later venous obstruction with hemorrhagic infraction of the left occipital lobe, and upper brain stem.

Doctor Aring: The discussions by the pathologists lends clarification to much that was unclear. I am certain, as Dr. Johansman said, that the diagnosis would have been less obscure had we had historical data that surely must have been available had anyone taken the trouble to look for them. Dr. Shaver's presentation leads us to believe that the patient must have had cerebral symptoms prior to those mentioned in the protocol. But these things happen; this is not a fairy story, rather the case presented an immediate problem with which doctors had to struggle although the immediacy obscured the cause. We should study cases such as this, inadequate though they be, because from them we learn.

Student: Is anyone willing to speculate on why the development of a cardiac lesion?

Doctor Johansman: The lesion appears to be that type which follows infectious fevers. I thought of diphtheric myocarditis; however, this is not definite. It would be helpful if the history contained some information allowing us to make such a diagnosis; in the stress of the emergency, no one obtained more than a brief outline of the story. It is certainly not a rheumatic myocarditis which may occur in an individual of this age.

Doctor Aring: Among the written discussions by the students there were two with the diagnosis of cerebral emboli; one considers the source to be infectious endocarditis, the other lung abscess. I would like to ask how the diagnosis of cerebral embolism was arrived at?

Doctor McIntyre: I think that if we had followed the first lead of vascular lesion occurring during sleep, then we would have made a more nearly correct diagnosis. In other words, those less encumbered with neurological information had the better chance of arriving at a correct diagnosis, the history being as inadequate as it was. Had an air injection been done, still it would have been thought that he had a tumor because of the tremendous shift of the lateral ventricles away from the largest lesion.

*Mr. Neal I. Aronson:** In studying the protocol, I came to the conclusion that the patient's inability to talk was due to a complete motor aphasia, the lesion being in or near the convolution of Broca. Realizing that my understanding of aphasia was slight, I consulted Nielsen's book, "Agnosia, Apraxia and Aphasia" and, quite by chance, came across the following paragraph: "As to the nature of the lesion it should be said that neoplasms rarely destroy the area immediately affected but rather push aside the structures. For this reason one need not expect to apply one's knowledge of aphasia for purposes of cerebral localization in cases of tumor. There are few exceptions. However, the usual lesion causing aphasia of Broca is vascular or traumatic. . . ." At this point, especially in view of the rather apoplectic nature of the onset which occurred in sleep, I began to think of a vascular accident as the responsible lesion in this case.

Except for a few transient attacks of syncope the month previous to admission, there was no evidence of symptoms referable to the cranium previous to the sudden onset, except perhaps for the inability to raise the eyes above the horizontal plane which I interpreted as a partial nuclear aplasia in the presence of other signs of congenital maldevelopment. It is conceivable that such a paralysis of upward gaze could be associated with a tumor of the pineal gland, tegmentum or thalamus, but there is little to support this in the way of signs. Besides, the signs, in my estimation, pointed mostly to a lesion in the dominant cerebral cortex.

Signs of pituitary dysfunction were present, but thought to be due to a multiple glandular dysfunction which was not thought to be remarkable in a patient underdeveloped from birth. There was no history of progressive visual difficulties, and the thinning of the posterior clinoids in the presence of a normal sella turcica was not considered to be significant.

Although the signs of increased intracranial

*Senior student.

pressure and the high protein content of the ventricular fluid were strong arguments in favor of a neoplastic process, the history was not thought to be, and therefore I did not allow myself to be too greatly influenced by a laboratory finding which according to all the literature mitigated against a diagnosis of vascular accident with clear cerebrospinal fluid.

Once I had made up my mind that a vascular disorder was a possibility I set about trying to decide what sort of vascular lesion it was. I immediately ruled out subarachnoid hemorrhage because of the water clear ventricular fluid. There was no reason to assume that an internal hydrocephalus existed and therefore, I concluded that there was free access to the subarachnoid space from the ventricular system.

Massive cerebral hemorrhage was ruled out on the basis of the fact that such a lesion involving the large area presumed to be affected in this case would be evident on trepanation and would undoubtedly have caused bloody ventricular fluid.

It then remained to decide between thrombosis and embolism. Both were considered as practical diagnoses on the basis of the onset and physical findings. The edematous brain was in keeping with vascular occlusion since edema and congestion are the after effects of the ischemia produced by sudden cutting off of blood flow. Another point which made such a diagnosis attractive was the distribution of the lesion. It was felt that the middle cerebral artery supplied a large portion of the area involved, by far the most common area affected by stroke. In Merritt's series of 62 cases of circumscribed lesions the area of the middle cerebral artery was involved in 47.

Since embolus may occur at any age and thrombosis is most often associated with old age, the former was considered to be a more likely choice. The temperature, respiratory rate and heart rate were in keeping with this diagnosis. The shock-like blood pressure was explained as being due to the terminal stage of the illness.

The signs of an irritative lesion of the cortex on the opposite side were believed to be due possibly to the edema of the affected side shifting the opposite cortex against unyielding skull. The signs of increased intracranial pressure were explained on the basis of the edema and congestion of the brain, as was the bilateral abducens palsy.

The picture viewed as a whole, seemed more characteristic of a vascular lesion than anything else, especially after being somewhat influenced by Nielsen's remarks. The one insurmountable obstacle seemed to be the high protein content of the intraventricular fluid. However, it was not altogether insurmountable. When you discussed ablumino-cytologic dissociation in relation to infectious polyneuritis, I got the impression that one should not rely too heavily on the laboratory to rule in or rule out a diagnosis, and that when the laboratory was at odds with the clinical picture, it is the former which is to be held in doubt. I felt that this situation pertained here and therefore my diagnosis of embolic phenomena.

Dr. Aring: An excellent attack; I myself was intrigued by the catastrophic onset, but perhaps even more so by the tremendous increase in ventricular protein. I had not known up to now that embolic phenomena in the brain resulted in a cerebrospinal fluid protein increase of this magnitude. Having seen Doctor Shaver's slides, I am able to envision such an occurrence with the multiplicity of lesions that he has shown us immediately adjacent to cerebrospinal fluid spaces.

I would like to say a word in behalf of the intelligent person untrained in a discipline. Unencumbered with the lumber of prepossession or with facts if you will, he is able in some cases to ignore the trees and to see the forest.* For this reason we may continue to expect with confidence that discoveries will come from without the fraternity. Therefore, it is most important that we handle with benevolence novitiates, crusaders, renegades, fanatics and any other characters whom we, in our wisdom, may consider odd and angular, even though benevolence be tinged with a healthy skepticism. Doctor Kurt Tschiassny has pointed to a quotation from Schiller's "Words of Faith" which bears on the point:

> "And that which the wise in his wisdom ne'er knew,
> Can be grasped by the mind that is childlike and true."

*See Colson, Z. W.: "The importance of being ignorant," *The Laryngoscope*, 57:683-689, 1947.

Psychodamnitis

RAYMOND HEADLEE, M.D.
Wauwatosa, Wisconsin

To the person caught in a web of speculative thinking it all seems logical, important and yet somehow hopeless. To the physician it seems a form of obsessive (or phobic) thinking. Perhaps then the entire concept of psychodamnitis, the meaning of which must be clear to those who have practiced psychiatry, is but a new dress for an old illness. Or it is something new, something which results from a substitution of words for realities on the part of both patient and doctor. If this autistic pastime were only a pastime, no harm would be done. But if the game of psychodamnitis holds a patient's progress at naught, or further complicates his chance for recovery, then it is high time some note be made.

Dr. Burlingame[1] mentions one form of this in his warning against overeducation of the public in things psychiatric. That a too-glib use of dynamic terms may serve first as an excuse for misbehavior, second as a smoke screen over actual illness, and third to drive patients to ill qualified personnel, is his contention; it has been echoed many times in both popular and scientific journals.

Dr. Freyhan[2] uses the term "psychiatrism" to denote this tendency. He refers to an oververbalization of dynamics as a "sad state of mechanized thinking," and further bemoans (with Dr. Braceland[3]), the extension of psychiatric thought into nearly every field of human endeavor.

At the risk, then, of being accused of further obscuring an already murky field of thought the author would like to present this new term, plus a few short illustrative cases, to illustrate the ill effects of psychodynamics. To separate this clearly from the valuable use of insight into psychological mechanisms, when properly used and timed, the author cannot help but coin *Psychodamnitis* (not, he hopes, after the fashion of Professor Fuddleton, in The Atlantic for June, 1948, page 87).

Case 1: College sophomore, male, age 20. Opening remarks were, "Doc, I've got an oedipus complex." First few interviews were full of complex mechanisms, most of which were meaningless; no consistent pattern of psychological thought could be made out. After about four interviews "treatment" was terminated since no real problem could be discovered.

Case 2: High school student, male, age 15. Both parents needed and were getting psychoanalysis elsewhere, sent boy in for preventive treatment. First interview was a masterpiece of pseudo-psychodynamic jargon, which included "reasons" for everything, including why psychiatrists chose that profession. This boy had some real inner tensions, although he managed to cover his insecurity and fears with a brave front (he was a large, athletic boy, handsome in fact). The problem was one of unlearning, rather than learning.

Case 3: A 38 year old housewife, with two children. By all informants she was perfectly normal and healthy until about 5 months before admission to Milwaukee Sanitarium. At this time she began to be somewhat depressed but still able to do her work. She started seeing a psychiatrist three times a week for "psychoanalysis." She saw him regularly for about four months, becoming steadily worse until hospitalization was necessary. She had obsessive ideas of guilt over her "narcissm and the false front she had for years." In telling of her treatment she said that she had "discovered that she had been a horribly self-centered person for all these years; perhaps suicide was the only way out." She was treated with CO_2 and became completely psychotic. After only two weeks of insulin coma treatments (with an unusually low number of actual comas) she recovered, began to wonder why she had ever had such "peculiar" thoughts; after another two or three weeks of convalescence she went home. She has now been well (and "unselfish") for about half a year.

Case 4: Entrepreneur (unsuccessful) male, age 35. Came in with complaint of stomach "ulcers," inability to eat any sort of fat

(except butter, of which he ate a quarter of a pound at each meal), and a general feeling of dissatisfaction with his own life. He had read all of the recently published psychiatric books, some of them twice. He handed me 200 dreams, which he had typed up, plus his own interpretations of them; he had also prepared 16 pages of single-spaced typing on which was printed his own outline (based on his reading) of the causative mechanisms of his present illness. It was impossible to help this man in any way.

Discussion

No attempt is made to present psychodamnitis as a disease in itself. It may be but a tendency, or a factor in a number of larger nosological entities. In view of the vast number of books, magazine articles, comic strips (and now even One Man's Family) which are dealing directly with psychiatric dynamics, it is necessary to face reality. Comments, criticisms and elaborations are invited.

Summary

Warning is sounded regarding the overemphasis of psychological mechanisms in human adjustment. To do so is to fall victim to *Psychodamnitis,* which, if not a disease in itself, is a factor to be considered in present day psychiatry. Four cases are presented in brief outline.

REFERENCES

1 Burlingame, C.C.: "One Hundred and Twenty-third Annual Report," *The Institute of Living,* 1947.

2 Freyhan, F. A.: "Psychiatric Realities," *J. Nerv. and Ment. Dis.,* 106:482, October 1947.

3 Braceland, F. J.: "Psychiatry and the Returning Veteran," *Ment. Hyg.,* 30:33, 1946.

Book Review

The Alcoholic Woman: By Benjamin Karpman, M.D., Washington, D. C.: *The Linacre Press,* 1948.

This book bears the subtitle "Case Studies in the Psychodynamics of Alcoholism," a phrase which summarizes its contents very well.

The entire book is concerned with three promiscuous women in whom the problem of alcoholism is secondary. The genesis for this combination of promiscuity and alcoholism, according to the author, lies in the insecurity and inferiority feelings engendered by stupid parents. The parental stupidity idea is very much emphasized in this connection.

Perhaps the most interesting as well as instructive part of this book is the dream analysis. Almost half its pages are devoted to the dream life and the author's interpretation of the dreams of each of these cases. The patients had been under his study for several years; consequently he had accumulated a wealth of material in this regard. He utilizes this information superbly in explaining their behavior and thinking patterns.

The reviewer recommends this book to those interested in the psychoanalytical approach to the problem of alcoholism.

—George Constant, M.D., Galveston, Texas.

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